Case Report

Sudden cardiac death following blunt chest trauma: commotio cordis

Robert J Douglas

Sportsmed SA, 32 Payneham Road, Stepney, South Australia, 5069

Corresponding Author: Robert Douglas, Email: rabs01@hotmail.com

BACKGROUND: There have been numerous reports of sudden cardiac death attributable to the condition of *commotio cordis*. Primarily, these are reports from the USA. Although three Australian cases have been mentioned in the published literature, the present case appears to be the first described Australian case.

METHODS: A man was brought to the Emergency Department after sudden collapse while playing cricket. His medical history was suggestive of hitting by a cricket ball while batting.

RESULTS: The epidemiology and mechanism of arrhythmia induction in *commotio cordis* are discussed. The emergency management of *commotio cordis* is outlined.

CONCLUSIONS: Commotio cordis is rare in sports (and Emergency Medicine). However it has a high mortality rate, and rapid recognition of the condition allows early defibrillation, generally with a good outcome. The improvement of participant care is recommended at community and other sport events.

KEY WORDS: Arrhythmia; Sudden cardiac death; Commotio cordis; Cricket

World J Emerg Med 2011;2(3):234-236
DOI: 10.5847/ wjem.j.1920-8642.2011.03.015

INTRODUCTION

There have been numerous reports of sudden cardiac death attributable to the condition of *commotio cordis*. Primarily, they are from the USA. Although three Australian cases have been mentioned in the literature, the present case appears to be the first one described in Australia.

CASE REPORT

A 33-year-old man in cardiac arrest was brought to the Emergency Department (ED). He had been playing cricket when he had fallen to the ground. It was assumed that he had been struck on the head by a ball and been rendered unconscious by the blow. An ambulance was called when he was found to be unresponsive. No firstaid action was taken by players, officials or spectators. On arrival of an ambulance crew to the scene he was unresponsive and pulseless with a Glasgow Coma Scale (GCS) of 3. A "rhythm strip" determined that he was in asystole. Cardio-pulmonary resuscitation (CPR) was given and he was intubated on-scene. On arrival to the emergency department, he was pulseless and unresponsive, and a three-lead monitor trace again demonstrated asystole. His pupils were fixed and dilated. No further resuscitation was undertaken, and he was pronounced to be "dead on arrival" (DOA).

Further history suggested that he may have been struck on the chest by the cricket ball immediately prior to his collapse. It was surmised that death was due to "commotio cordis".

RESULTS

Epidemiology of commotio cordis

Commotio cordis, is a term derived from the Latin

for "agitated heart".^[1] The condition was first described in 1876, when it was reported that a porter had died when the yoke he had been pulling "snapped" and he had fallen chest-first onto the ground.^[2]

Sudden cardiac death (SCD) in athletes is very rare in Australia. It appears to be more common in the United States of America, most likely due to significant differences in the participation rates in sports which are known to have an increased risk for injury-related death (eg baseball, lacrosse). Maron^[3] estimated that SCD in young athletes of the USA occurs at a rate of 1/200 000 high school athletes per academic year. In an analysis of 1866 cases of SCD amongst young athletes, SCD due to commotio cordis was found in 65 (3%) of reported cases. [4] Using data from the US commotio cordis Registry (up to September 1st, 2001) Maron et al^[5] identified 128 confirmed cases of commotio cordis. Of these cases, 122 (95%) were male, with a mean age of 13.6±8.2 years (median 14 years, range 3 months to 45 years). About 78% of the cases were less than 18 years old, and one third were less than 10 years old. Most cases (79/128 cases [62%]) were seen during organised sport events. It has been suggested that commotio cordis occurs more commonly in the "younger" population as they have a narrow, pliable chest wall that facilitates transmission of energy from the site of impact to the myocardium.^[6]

Death due to *commotio cordis* has been found to be more common in lacrosse (0.63 deaths per 100 000 person-years) than in other sports (with the exception of (ice) hockey), despite the overall mortality of lacrosse (1.46 deaths per 100 000 person-years) being similar to that of other competitive body-contact (US) sports.^[7]

Death due to *commotio cordis* has been reported as the result of a precordial blow from numerous objects including: ice hockey puck, lacrosse ball, cricket ball, soccer ball, baseball, softball, and snowball.

It also occurs as a result of tackles in rugby and American football.^[5,8] There are also some more unusual causes, such as parents disciplining children (5 cases), "play-boxing" with a friend (6 cases), and "struck in the chest by a friendly dog" (1 case). [8] The survival rate of *commotio cordis* events has been only 15%, which is attributed to delays in the commencement of CPR. [6]

Mechanism of arrhythmia induction

Experiments on a swine model have shown the pathophysiology of *commotio cordis* or an impact on cardiac silhouette is necessary to induce *commotio cordis*. ^[9] The impact causes an increase of the peak left ventricular (LV) pressure, which has been determined

to correspond directly to the risk of induction of VF. It is necessary for the impact to occur within a "window" of 30-15 millisconds (msec) prior to the appearance of T-wave peak on ECG. [10] Impacts obeying these parameters cause a selective disruption of the myocyte cytoskeleton [11] which in turn activates selective K(+) (ATP) channels. [12] These events interfere with ventricular repolarization, resulting in a premature ventricular contraction, and ultimately, VF.

Other experiments have shown that there is a relationship between the velocity of impact of the projectile with the chest and the risk of induction of *commotio cordis*. Impact at less than 20 miles per hour (mph) (32 kilometres per hour) is unable to induce VF. The VF induction rate increases incrementally from 7% at 25mph (40 kph) to 68% at 40mph (64kph). The induction rate then diminishes at above 50 mph (80 kph). It has been determined too that the peak LV pressure is related in a similar Gaussian manner.^[13]

Emergency management of commotio cordis

The simple answer is that the condition should be quickly recognized and not subject to misdiagnosis, such as occurred in this case. Early recognition of *commotio* cordis allows the rapid implementation of bystander CPR, maintained until external defibrillation can be achieved. There are reports on successful resuscitation following early recognition of VF. Ngai et al [14] reported the case of a teenager struck in the chest while playing rugby. Bystander CPR was implemented, and VF was noted on rhythm strip. External defibrillation was activated, and a successful return to sinus rhythm achieved. The patient "recovered well neurologically." Salib et al^[15] reported a successful resuscitation of a child who was struck while playing baseball. Unfortunately, not all cases are successful, as Maron et al^[16] reported in the case of a young athlete who received prompt defibrillation but could not be resuscitated.

CONCLUSION

Commotio cordis is a rare form of SCD in young athletes; however should it occur there is a very low rate of survival. It is a disorder that is exceedingly rare in Australia, and although previously reported to have occurred in Australia^[17] this appears to be the first described Australian case. The induction of commotio cordis relies upon an object striking the chest over the cardiac silhouette during the vulnerable period of ventricular repolarization, on the upslope of the T-wave.

The induction is further dependent on the speed of the object striking the chest, and by extension, the energy of impact of the object with the chest. Rapid recognition of the signs and symptoms of *commotio cordis* with the immediate implementation of CPR (and external defibrillation, if available) can reduce the mortality rate of the condition.

It is recommended that in all cases of collapse during a sport event an assessment of circulation be made by first responders, and CPR be implemented should the pulse be absent. Training of sports officials in the basics of life support and CPR as well as the presence of an automated external defibrillator should be required for all sport events where an episode of *commotio cordis* is possible.

ACKNOWLEDGEMENTS

My thanks go to Dr. Tonia Mezzini for critical appraisal of the manuscript.

Funding: No external funding provided.

Ethical approval: Not required.

Conflicts of interest: No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

Contributors: Paper conceived and written by Douglas R.

REFERENCES

- Morwood James. The pocket oxford latin dictionary. Oxford University Press, 1994.
- 2 Nelaton A. Elements de pathologie chirurgicale. Librairie Germer Bateliere, Paris, 1876. (Cited from Kohl P (1999). Commotio cordis: early observations (Letter). Heart 82: 397.
- 3 Maron BJ. Cardiovascular risks to young persons on the athletic field. Ann Intern Med 1998; 129: 379-386.
- 4 Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation 2009; 119:1085-1092.
- 5 Maron BJ, Gohman TE, Kyle SB Estes NA 3rd, Link MS.

- Clinical profile and spectrum of commotio cordis. JAMA 2002; 287: 1142-1146.
- 6 Maron BJ, Estes NA 3rd, Link MS. Task Force 11: Commotio cordis. J Am Coll Cardiol 2005; 45: 1371-1373;
- 7 Maron BJ, Doerer JJ, Haas TS, Estes NA, Hodges JS, Link MS. Commotio cordis and the epidemiology of sudden death in competitive lacrosse. Pediatrics 2009; 124: 966-971;
- 8 Fox D. Lethal Impact. New Scientist 2003; 2410: 38-39.
- 9 Link MS, Maron BJ, VanderBrink BA, Takeuchi M, Pandian NG, Wang PJ, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. J Am Coll Cardiol 2001; 37: 649-654.
- 10 Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, Lee MY, et al. An experimental model of sudden death due to low-energy chest-wall impact. N Engl J Med 1998; 338: 180-181.
- 11 Madias C, Maron BJ, Supron S, Estes NA 3rd, Link MS. Cell membrane stretch and chest blow-induced ventricular fibrillation: commotio cordis. J Cardiovasc Electrophysiol 2008; 19:1304-1309.
- 12 Link MS, Wang PJ, VanderBrink BA, Avelar E, Pandian NG, Maron BJ, et al. Selective activation of the K(+) (ATP) channel is a mechanism by which sudden death is produced by low-energy chest-wall impact (Commotio cordis). Circulation 1999; 100: 413-418.
- 13 Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zhu W, Estes NA 3rd. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). J Am Coll Cardiol 2003; 41: 99-104.
- 14 Ngai KY, Chan HY, Ng F. A patient with commotio cordis successfully resuscitated by bystander cardiopulmonary resuscitation and automated external defibrillation. Hong Kong Med J 2010; 16: 403-405.
- 15 Salib EA, Cyran SE, Cilley RE, Maron BJ, Thomas NJ. Efficacy of bystander cardiopulmonary resuscitation and out-of-hospital automated external defibrillation as life-saving therapy in commotio cordis. J Pediatr 2005; 147: 863-866.
- 16 Maron BJ, Wentzel DC, Zenovich AG, Estes NA 3rd, Link MS. Death in a young athlete due to commotio cordis despite prompt external defibrillation. Heart Rhythm 2005; 2: 991-993.
- 17 McCrory PR, Berkovic SF, Cordner SM. Deaths due to brain injury among footballers in Victoria, 1968-1999. Med J Aust 2005; 172: 217-219.

Received March 20, 2011 Accepted after revision June 26, 2011